of mortality and hospital stay. This result is misleading. Should we now offer RPM to the millions of heart failure outpatients? The most recent trials (2,3) failed to demonstrate convincing benefits in these end points. What caused the discrepancy? In the era of good baseline medication, growing defibrillator implantation rates, scheduled visits, and good self care, it is essential to identify the patients who might benefit from additional RPM and also those who will not. What are the determinants of outpatient responsiveness? When should RPM be used and for how long? What systems are most suitable? What makes interventions effective? Apart from the diversity of healthcare delivery systems requiring coordination in each country, there might be disease-related determinants of receptivity to RPM. The efforts of future trials should focus on these aspects. The challenge is to identify patients who require daily contact with healthcare experts as well as those who can continue to receive usual care without harm. A very smart technology calls for very intelligent clinical implementation.

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## Reply

We thank Drs. Winkler and Koehler for their interest in our report (1) regarding the assessment of effect of remote patient monitoring on the outcome of chronic heart failure patients. We appreciated their provocative thoughts about unmet needs in structured disease management program (e.g., identification of the patients who most likely benefit from the technology, determinants of outpatient responsiveness, what makes interventions effective), which we all share; however, we are afraid that none of these questions might have found an answer, given the lack of published data. As far as the differences in outcome of 2 of the most recently published studies and the results of our meta-analysis are concerned, we believe that they are much less than what Drs. Winkler and Koehler perceived.

The study by Mortara et al. (2) showed a similar outcome between usual care and remote monitoring (indicated in the study as home telemonitoring). However, patients in the Mortara et al. study were at least 5 to 10 years younger than those included in our meta-analysis, and they were in a much lower New York Heart Association (NYHA) functional class (ranging from 34% to 49% in NYHA functional class >3 compared with 54% [randomized controlled trials] and 83% [observation cohorts] in our meta-analysis). Moreover, there was an unexplained imbalance, as already emphasized by Mortara et al., in baseline characteristic in the large Polish cohort as indicated by a more advanced NYHA functional class, significantly lower left ventricular ejection fraction, higher dyspnea score, and much lower sodium plasma level for those patients assigned to home telemonitoring. A post hoc analysis revealed a highly significant interaction between home telemonitoring and country in the association with the number of hospital stays ($p = 0.004$) and in the combined end point of cardiac death and heart failure hospital stay ($p = 0.004$). If one would put in perspective the outcome of the Italian cohort of the study by Mortara et al. with the results of our meta-analysis, an impressive similar benefit of remote monitoring compared with usual care would be found.

The study by Dar et al. (3) was a small, prospective, randomized controlled study including 182 patients randomized to usual care versus home monitoring. Although the baseline demographic characteristics of these patients were similar to those reported in the studies included in our meta-analysis, only 74 patients in the home monitoring arm and 79 patients in the usual care completed 180-day follow-up. Thus, the relative weight of the study by Dar et al. (3) in our meta-analysis would be relatively low, and importantly, approximately 50% of the studies we meta-analyzed had a similar duration of follow-up. Of note, there were 14 deaths in the home monitoring arm and only 4 deaths in the usual care group. Overall, this was an extremely high death rate for a very short follow-up but also impressively different between 2 treatments. We were not able to find any comparative study in our meta-analysis reporting similar death rates, which let us question about the reasons (not addressed in the study). Indeed, the death rate at 12 months in all randomized controlled studies we meta-analyzed ranged from 14.1% (95% confidence interval [CI]: 12.8 to 15.4) to 11.7% (95% CI: 10.7 to 12.9) in the usual care and home monitoring arm, respectively, and in observational studies it ranged from 13.0% (95% CI: 10.9 to 15.3) to 6.8% (95% CI: 5.3 to 8.6).

In conclusion, there is no doubt that the studies by Mortara et al. (2) and Dar et al. (3) both represent important contributions to implementation of remote monitoring. Their relative weight needs to be defined in future meta-analysis, keeping in mind some important methodological issues of each study.

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# Not Just Coronary Arteritis, Kawasaki Disease Is a Myocarditis, Too

There is a great deal of insight in the points of view addressed recently by Gordon et al. (1) and by Gersony (2). One could not but notice an unbalanced aspect in the commentary article, however. This has nothing to do with the respected opinion of Dr. Gersony; on the contrary, it has to do with the general misconception connecting Kawasaki disease (KD) to merely 1 fact: the related coronary artery complications. The original editorial article by Gordon et al. (1) is intended to convey a message to the Adult Cardiology Society urging for an educated awareness of the perceived impact of the KD-related cardiovascular disturbances beyond childhood. Whether some aspects of the disease gained the deserved emphasis in the original review article or not, the rebuttal commentary was completely distracted from essential realities associated with KD. Although Gordon et al. (1) identified the often-missed importance of the consequences of KD on the myocardium, Gersony (2) limited his discussion to the sole coronary artery complications of the disease. That is exactly the problem. In my mind, it is important to put a brake on the problematic coronary aneurysms, be it only for a minute. The hidden face of the moon in this disease, the myocarditis, must not be underestimated (3) This myocarditis is evidenced by serial myocardial biopsy studies from patients without coronary aneurysms (4,5). It is also suggested by echocardiography studies (6) and by biochemical markers reflecting the myocardial response to the inflammatory process upon the onset of the disease (7). Four decades since the initial recognition of KD as a separate entity from resembling ailments have not permitted researchers to uncover its etiology. The inflammatory involvement of the myocardium and its long-term consequences deserve a serious look and a methodological follow-up.

The incidence of KD is on the rise, not only because of the modified diagnostic paradigm, which encourages the diagnosis of cases with incomplete clinical criteria (8,9), but also because of the recent awareness of the diagnosis, not only in North America but also in the most populous countries of the globe as well (10,11). It is appropriate and wise to inform KD patients that there are insufficient data to adequately calculate their cardiovascular prognosis, with the exception of the minority who sustained a severe coronary artery injury. Should we wish to care for the remaining 99% of KD patients, the myocarditis trail—not just that of the coronary arteries—needs to be followed. And as Gersony (2) correctly concludes, the American Heart Association guidelines are simply guidelines, subject to continuous updates.

Now, back to the injured coronary arteries, resolved or unresolved. If making patients aware of their antecedent KD diagnosis as a potential cardiovascular risk factor deeply affects their psyche, then physicians must also refrain from discussing obesity, for example, when counseling primary or secondary prevention of cardiovascular diseases.

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## Reply

I thank Dr. Dahdah for his interest in my paper (1). In his letter to the editor, Dr. Dahdah raises concerns about the long-term effects of myocarditis on adults who had Kawasaki disease (KD) as a child, and that my commentary, which pertained to the risk of late coronary artery events, did not address this potential issue. As he indicates, a myocardial inflammatory process in the acute phase of KD has been well documented, even when coronary involvement may have been minimal or even absent. However, Dr. Dahdah's assertion that late manifestations of acute myocarditis are likely to be a serious threat to the adult who had KD is not evidence based. The references accompanying his letter do not describe a single case of an adult with late myocarditis or nonischemic cardiomyopathy, and there have been hundreds of thousands of patients who have had KD. Furthermore, the biopsy studies carried out in 1978 and 1981 were obtained from patients in the acute and subacute phases of the disease. By no means can